ventricular volume overload or left atrial volume overload, and to the morphologic characteristics of the mitral valve (i.e., simple mitral valve prolapse vs. flail leaflet or the presence of severe myxomatous infiltration), cannot yet be analyzed. Recently, it has been suggested that mitral valve prolapse is uniformly a good prognosis (3). It is unclear how these various components of the clinical presentation of patients with mitral valve diseases contribute to the outcome, in particular to the risk of sudden death. We agree that further studies are needed on the outcome of various types of mitral valve disease. Such studies will require analysis of large population-based groups of patients with mitral valve disease. At this point, our study allows us to define the notable risk of sudden death incurred under conservative management by patients with flail leaflets, who represent a large group of candidates for surgical correction of mitral regurgitation.

Maurice Enriquez-Sarano, MD, FACC
Cardiovascular Diseases and Internal Medicine
Mayo Clinic
200 First Street SW
Rochester, Minnesota 55905

PII S0735-1097(00)00953-0

REFERENCES

1. Grigioni F, Enriquez-Sarano M, Ling L, et al. Sudden death in mitral regurgitation due to flail leaflet. J Am Coll Cardiol 1999;34:2078–85.
2. Kannel WB, Doyle JT, McNamara PM, Quickenton P, Gordon T. Precursors of sudden coronary death: factors related to the incidence of sudden death. Circulation 1975;51:606–13.
3. Freed LA, Levy D, Levine RA, et al. Prevalence and clinical outcome of mitral-valve prolapse. N Engl J Med 1999;341:1–7.

U-Wave Alterations: Singular Noninvasive Electrocardiographic Diagnostic Markers

The recent absorbing report by Miwa et al. (1) is, secondarily, a reaffirmation of the considerable utility of the 12-lead electrocardiogram (ECG) and why it continues to be the most frequently used cardiovascular laboratory procedure. It was refreshing to learn that exercise-induced prominent U waves implicated significant left circumflex or right coronary artery disease and that patients with good collateral vessels could be identified by the finding of exercise-induced U-wave alterations. Furthermore, these alterations predicted the development of acute myocardial infarction or hemodynamic instability for low-risk patients upon abrupt closure of a stenotic coronary artery during coronary angioplasty.

There are three practical tenets regarding the at-rest negative U wave on the ECG. There are diverse cardiovascular etiologies, and only with knowledge of the history and physical findings will the full cognitive base residing in the experienced electrocardiographer's repertoire be mobilized. It is an extremely important wave, as it may be the earliest and only marker of an evolving myocardial infarction (2), and it is an important clue in identifying the congenital long QT syndromes, such as the Jervell and Lange-Nielsen and the Romano-Ward syndromes, which harbor a malignant arrhythmogenic potential (3). Negative U waves may also be recorded in the presence of left ventricular enlargement; left anterior descending coronary artery disease (4); valvular heart disease such as aortic stenosis, mitral regurgitation and aortic insufficiency; and hypertension and variant angina (5). Second, transient U-wave inversion may be seen with both hypertension and variant angina; it can be differentiated on the ECG by initial or terminal negative deflections within the TP segment—the latter as related to myocardial ischemia (5). Finally, Miwa et al. (1) found a lower ejection fraction in patients with severe angina, exercise-induced U-wave alterations and good collateral vessels. Interestingly, in this vein, some 20 years ago, Gerson and McHenry (4) reported that at-rest U-wave inversion was an indicator of stenosis of the left anterior descending coronary artery, and they also found that U-wave negativity was a significant predictor of left ventricular dysfunction, usually segmental anteroapical akinesia or dyskinesia. Miwa et al. (1) and Gerson and McHenry (4) are applauded.

John A. M. Morphet, MD, FRCP(C), FACC, FESC
R.R. #1
225 Breisacher Road
Huntsville, Ontario
Canada P1H 2J2

PII S0735-1097(00)00937-2

REFERENCES

1. Miwa K, Nakagawa K, Hirai T, Inoue H. Exercise-induced U-wave alterations as a marker of well-developed and well-functioning collateral vessels in patients with effort angina. J Am Coll Cardiol 2000;35:757–63.
2. Fisch C. The clinical electrocardiogram: sensitivity and specificity. ACC Curr J Rev 1997;6:71–5.
3. Moss AJ, Schwartz PJ. Delayed repolarization (QT or QTU prolongation) and malignant ventricular arrhythmias. Mod Concepts Cardiovasc Dis 1982;51:85–90.
4. Gerson MC, McHenry PL. Resting U-wave inversion as a marker of stenosis of the left anterior descending coronary artery. Am J Med 1980;69:545–50.
5. Miwa K, Miyagi Y, Fujita M, Fujiki A, Sasayama S. Transient terminal U-wave inversion as a more specific marker for myocardial ischemia. Am Heart J 1993;125:981–6.